A study in the *Journal of Biological Chemistry* identifies a new therapeutic approach to treat prostate cancer.

Conducted at Cincinnati Children's Hospital Medical Center, the research shows that expression of the FoxM1 protein is essential for prostate cancer to develop in mouse models. The study appears in the journal's Aug. 2 edition.

The study also shows that depletion of FoxM1 in prostate epithelial cells inhibits tumor cell proliferation, the process by which new blood vessels are formed, and metastasis—the spread of cancer to other organs of the body.

"It is possible that FoxM1 is important for both cancer initiation and cancer progression," says Tanya Kalin, MD, PhD, a physician-scientist in the division of Pulmonary Biology at Cincinnati Children's and senior author of the study. "Our findings provide the foundation for the development of new therapeutic approaches based on inhibition of FoxM1."

FoxM1 is known to be involved in most solid tumor cancers. Kalin's lab published a study in 2006 showing that increased levels of FoxM1 were associated with the development and progression of prostate cancer in mice. Although the current study focuses primarily on prostate cancer, the findings could also help researchers better understand the pathogenesis of pediatric disease.
In this new study, Kalin and colleagues at Cincinnati Children's studied a novel mouse model of prostate cancer. In this model, loss of FoxM1 decreased tumor growth and metastasis. Without this model, over-expression of FoxM1, either alone or in combination with inhibition of a tumor suppressor known as p19ARF caused a robust proliferation of epithelial cells. This proliferation of cells was not enough to induce progression from proliferation to actual prostate cancer, but it caused small prostate epithelial tubes to shrink.

Provided by Cincinnati Children's Hospital Medical Center

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